AMENDMENTS TO THE CLAIMS

Claim 1 (Previously presented): A method of screening or validating an antiestrogen, said method comprising screening a test compound for the ability to activate transcription through an indirect estrogen response, the method comprising:

a) providing a cell comprising AP1 proteins, an estrogen receptor and a promoter comprising an AP1 site which regulates expression of a reporter gene;

b) contacting the cell with the test compound; and

c) detecting the expression of the reporter gene, wherein enhanced expression of the reporter gene indicates that said test compound has the ability to activate transcription through an indirect estrogen response and is not fully antiestrogenic.

Claim 2 (Previously presented): The method of claim 1, wherein the cell is an Ishikawa cell.

Claim 3 (Previously presented): The method f claim 1, wherein the cell over-expresses the estrogen receptor.

Claim 4 (Previously presented): The method of claim 1, wherein the promoter is genetically engineered to compr8ise an AP1 site.

Claim 5 (Previously presented): The method of claim 1, wherein the test compound is known to have antiestrogenic activity.

Claim 6 (Previously presented): The method of claim 1, wherein the cell is derived from uterine tissue.

Claim 7 (Previously presented): The method of claim 6, wherein the cell is a HeLa cell or an Ishikawa cell.

Claim 8 (Previously presented): A method of claim 1, further comprising the steps of:

a) providing a second cell comprising an estrogen receptor and a promoter comprising a standard estrogen response element which regulates expression of a second reporter gene;

b) contacting the second cell with the test compound; and

c) detecting the expression of the second reporter gene.

Claim 9 (Previously presented): A method of claim 8, wherein the response element is from thse *Xenopus* vitellogenin A2 gene.

Claim 10 (Previously presented): A method of claim 1, wherein the cell further comprises a promoter comprising a standard estrogen response element which regulates expression of a second reporter gene.

Claim 11 (Previously presented): A method of claim 10, wherein the response element is from the *Xenopus vitellogenin* A2 gene.

Claim 12 (Canceled).

Claim 13 (Previously presented): A method of screening or validating an antiestrogen, said method comprising screening a test compound for the ability to inhibit transcription through an indirect estrogen response, the method comprising:

a) providing a cell comprising AP1 proteins, an estrogen receptor and a promoter comprising an AP1 site which regulates expression of a reporter gene;

b) contacting the cell with the test compound and a compound known to mediate an indirect estrogen response;

c) detecting the expression of the reporter gene, wherein inhibition of expression of said reporter gene produced by said compound known to mediate an indirect estrogen response indicates that said test compound inhibits transcription through an indirect estrogen response and is a candidate antiestrogen.

Claim 14 (Previously presented): The method of claim 13, wherein the compound **[is]** known to mediate an indirect estrogen response is tamoxifen.

Claim 15 (Previously presented): A method of claim 13, wherein the cell over-expresses the estrogen receptor.

Claim 15 (Previously presented): The method of claim 13, wherein the promoter is genetically engineered to comprise an AP1 site.

Claim 17 (Canceled).

Claim 16 (Previously presented): A method for screening a test environmental compound for estrogenic activity mediated through an indirect estrogen response, the method comprising:

a) providing a cell comprising AP1 proteins, an estrogen receptor and a promoter comprising an AP1 site which regulates the expression of a reporter gene;

b) contacting the cell with the test compound; and

c) detecting the expression of the reporter gene, wherein enhanced expression of the reporter gene indicates that said environmental compound has estrogenic activity.

Claim 17 (Previously presented): The method of claim 16, wherein the cell further comprises a promoter comprising an estrogen response element (ERE) which regulates expression of a second reporter gene.

Claim 18 (Previously presented): The method of claim 16, where the reporter gene is CVAT.

Claim 19 (Previously presented): The method of claim 16, wherein the cell over-expresses the estrogen receptor.

Claim 20 Previously presented): The method of claim 16, wherein the cell is an ERC1 cell.

Claims 23-29 (Canceled).